Diving Hazards Unmasked

Estimating Infection Risk from Pathogen Exposure

Most recreational water quality standards are aimed at protecting beachgoers against accidental ingestion of or skin contact with water contaminated by fecal material. But the increased popularity of water sports such as kayaking, surfing, and diving, which often occur far from regulated bathing beaches, raises the question of the water-related health risks these sports entail. Now two researchers at the National Institute of Public Health and the Environment in the Netherlands have attempted to answer this question for divers [***EHP* 114:712–717; Schijven and Husman**]. The study is the first to establish estimates of how much water divers swallow, figures that can be used in calculating health risks involved with waterborne pathogen exposure during diving.

What lies beneath. A new study estimates divers' risk of developing infections when diving in fecal-contaminated waters.

The researchers used detailed questionnaires to ask occupational and sport divers about the number and duration of dives they made in ocean, coastal, and freshwater areas; whether a known pollution source was nearby; the type of diving mask worn (which affects the amount of water swallowed); and the amount of water typically swallowed per dive. Five equivalents enabled divers to estimate how much water they swallowed: nothing, a few drops (an average of 2.75 milliliters [mL]), a shot glass (25 mL), a coffee cup (100 mL), or a soda glass (190 mL). The questionnaire also asked respondents to detail past health complaints, including diarrhea, vomiting, nausea, and eye, skin, and ear problems.

Then the researchers calculated the risk of infection per dive and per year based on the volume of swallowed water reported and pathogen concentrations. *Campylobacter jejuni* and enteroviruses were used for the analysis; concentrations for these organisms in different kinds of surface waters were taken from the literature, and concentration distributions constructed.

The infection risks for *C. jejuni* were generally an order of magnitude higher than those for enteroviruses. For occupational divers, the greatest per-dive mean risk of infection was calculated at 2.8% in coastal waters near a sewage discharge. For sport divers wearing ordinary diving masks, the greatest mean risk was seen in freshwater recreational waters, where there was a 1.5% per-dive risk and a 25% per-year risk of getting an infection. The risk was about 10 times lower when sport divers wore full face masks.

Although occupational divers usually have the protection of a full face mask or diving helmet, they are far more likely than sport divers to dive in contaminated conditions—for example, to assess damage to underwater sewage pipes. They also tend to stay underwater longer. Thus, the chance for exposure goes up.

These relatively high infection risks may explain why 80% of the divers surveyed reported at least one of the health complaints listed on the questionnaire during the course of one year. The authors recommend that divers wear full face masks or helmets when diving in potentially contaminated waters, and that they stay informed about fecal contamination in diving areas. **–Nancy Bazilchuk**

A Fine Differentiation

Chlorpyrifos and Neuronal Development

Although chlorpyrifos has been restricted for use in the home, it is still permitted for agriculture and remains the most widely used organophosphate pesticide in the world. Animal studies and *in vitro* models have indicated that chlorpyrifos has direct and indirect effects on fetal and neonatal neural cell replication and differentiation. These effects include cholinesterase inhibition as well as immediate and delayed-onset changes in synapse formation, neurotransmitter release, neurotransmitter receptor expression, and intracellular signaling. Moreover, chlorpyrifos can exert simultaneous, opposite effects on axonal and dendritic growth. Now researchers from Duke University Medical Center show that chlorpyrifos has direct effects on the differentiation that determines the phenotypic fate of developing neuronotypic cells [***EHP* 114:667–672; Jameson et al.**].

One problem with *in vivo* animal studies is the difficulty of teasing out the indirect effects mediated by mother–fetus or mother–neonate interactions, as opposed to the direct effects of chlorpyrifos on the developing brain. Accordingly, attention has increasingly come to focus on *in vitro* models that simulate the development of two basic types of brain cells, neurons and glia.

The Duke researchers set up such a model using PC12 cells, a tumor cell line that originates from a neuronal phenotype and that can recapitulate all the major phases of neurodevelopment thought to be targets for chlorpyrifos. With the addition of the peptide known as nerve growth factor, differentiation begins: PC12 cells cease dividing and develop the characteristics of neurons, including axonal projections and specialization into either cholinergic or catecholaminergic transmitter systems.

Cholinergic systems have shown immediate and lasting damage when exposure to chlorpyrifos occurs during periods of rapid cell replication (when the neuronal cells are dividing) and differentiation. In contrast, chlorpyrifos exposure initially enhances the development of catecholaminergic systems, increasing the expression of the proteins characteristic of this system and enhancing synaptic activity;

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nevertheless, long-term brain function deficits eventually appear, mainly in the form of disruption of synaptic connectivity. The current study was aimed at answering three basic questions about *in vitro* exposure to chlorpyrifos: Does chlorpyrifos alter the ability of developing neurons to express a specific neurotransmitter phenotype? If so, at what stage of cell maturation does this occur? And do such changes occur at chlorpyrifos concentrations below those that affect cell viability?

The researchers evaluated PC12 cells in the undifferentiated state, at the initiation of differentiation, and at mid-differentiation. They contrasted the effects on cell viability, DNA synthesis associated with cell replication, and increased expression of enzyme markers that characterize cholinergic or catecholaminergic phenotypes: choline acetyltranferase (ChAT) and tyrosine hydroxylase (TH), respectively.

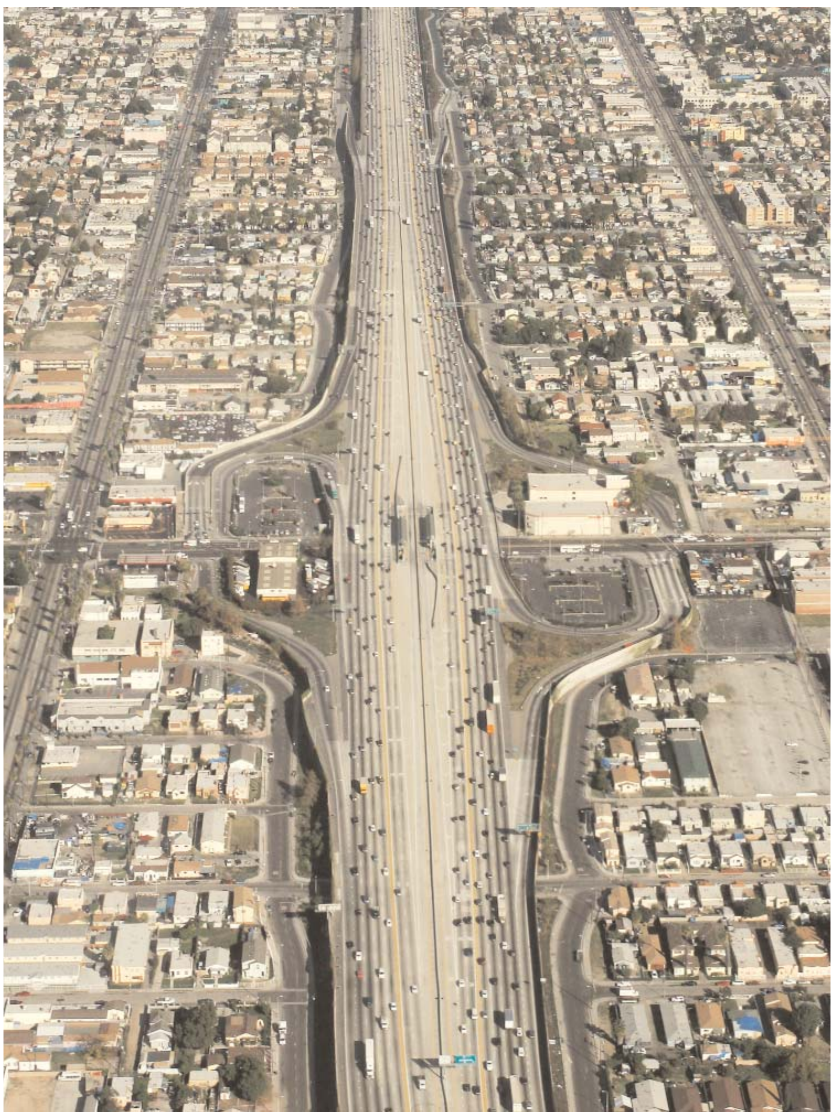
Chlorpyrifos exposure at the start of differentiation significantly reduced ChAT but not TH activity. With chlorpyrifos addition during mid-differentiation (four days after nerve growth factor pretreatment), ChAT was unaffected, but TH was increased slightly. Chlorpyrifos reduced DNA synthesis in the undifferentiated state, thereby impairing general neuronal cell development, whereas at the start of differentiation, it specifically impeded development of the cholinergic phenotype.

Chlorpyrifos administration *in vivo* is known to cause deficits in the number of neurons and cholinergic function. Because the researchers were able to reproduce these effects reliably *in vitro*, they suggest that chlorpyrifos directly influences the phenotypic fate of neuronal precursors. In addition, they suggest that their cell culture model could become useful for the rapid screening of neurodevelopmental outcomes with related, or even disparate, neurotoxicants. **–Julian Josephson**

The Freeway Running through the Yard

Traffic Exhaust and Asthma in Children

Since its inception 13 years ago, the Children's Health Study has indicated that air pollution in Southern California communities reduces lung growth and development, raises the risk of developing asthma, and increases school absences due to respiratory illnesses. The latest finding from the study team zeroes in on the impact of exposure to traffic-related pollutants at home, and shows that kindergarten and first-grade students who lived near busy roads experienced a higher prevalence of asthma [***EHP* 114:766–772; McConnell et al.**].



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Life on the streets. Children who live within a block of major roads are one and a half times more likely to report asthma or wheezing than those living four or more blocks away.

The researchers evaluated the respiratory health of 5,341 children relative to the distance that they lived from major roads, including highways, arterial roads, and freeways. The children, aged 5 to 7 years, lived in 13 communities. The team used detailed information about roadway type and traffic volume collected by the California Department of Transportation to develop a proxy for fresh traffic exhaust—the gases given off immediately around cars—at each child's home.

Children who lived within 75 meters of a major road (about the length of a city block) were approximately 1.5 times more likely to report asthma or wheezing compared to those living 300 meters or more from a major road. Among children with no parental history of asthma, those who had resided at an address close to heavy traffic since before age 2 experienced even higher risks (2.5-fold for asthma and 2.7-fold for wheezing), suggesting that a cumulative lifetime exposure to traffic pollutants may raise health risks. Girls showed a greater association between living near a major road and the health outcomes measured, for unknown reasons.

Few studies in the United States have looked at the connection between traffic and the prevalence of childhood asthma, but the results are consistent with emerging evidence from European studies. Smog and other regional pollution is slowly being brought under control by legislation. However, traffic exhaust represents a form of local pollution with public health consequences that is largely unregulated. As a start toward curbing the effects of exhaust, California recently passed a law that prohibits the construction of new schools within 500 feet of freeways. Locating playgrounds, parks, and sports fields a safe distance from busy roads may be another way to prevent children from inhaling exhaust fumes. **–Carol Potera**